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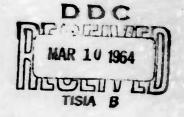
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TECHNICS FOR MEASUREMENT OF INTRAPLEURAL AND PERICARDIAL PRESSURES IN DOGS STUDIED WITHOUT THORACOTOMY AND METHODS FOR THEIR APPLICATION TO STUDY OF INTRATHORACIC PRESSURE RELATIONSHIPS DURING EXPOSURE TO FORWARD ACCELERATION $(+G_x)$

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(Prepared under Contract No. AF 33(657)-8899 by
E. H. Wood, A. C. Nolan, D. E. Donald, A. C. Edmundowicz and
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FOREMORD

The study on which this report is based was accomplished in the cardiovascular and human centrifuge laboratory of the Mayo Foundation, Mayo Clinic, Rochester, Minnesota, under the direction of Dr. Earl Wood under Air Force Contract No. AF 33(657)-8899, Project No. 7222, "Bio-physics of Flight," and NASA Contract R-43. Dr. Alvin S. Hyde, Multienvironment Division, Biophysics Laboratory, 6570th Aerospace Medical Research Laboratories, was the contract monitor. Dr. Wood was assisted in this study by Drs. A. C. Nolan, D. E. Donald, A. C. Edmundowicz and H. W. Marshall and Mr. W. F. Sutterer of the Mayo Clinic. Work on this project started 1 December 1961 and continued until 30 November 1962.

This study was made possible by the unstinting cooperation of many of our technical and professional colleagues in the Section of Physiology and Section of Engineering; among these Donald Hegland, Miss Lucille Cronin, Julius Zarins, William Hoffman, Robert Hanson and Mrs. Jean Frank are deserving of particular mention.

ABSTRACT

Pleural pressures were recorded simultaneously from the ventral and dorsal regions of the thorax using fluid-filled catheters inserted through the chest wall via No. 16 needles using an air-tight technic. Pressures were referenced to the catheter tip levels determined by A-P and lateral roentgenograms taken prior to and after a series of 1 to 3 minute exposures of 8 anesthetized dogs to accelerations of 2, 4 and $6G_X$ (supine horizontal and 15° head-up and head-down positions).

The negativity of intrapleural pressure in the ventral thorax was uniformly increased during exposures while intrapleural pressure in the dorsal thorax became positive. These changes are believed to result from the increase in weight of the lungs and other intrathoracic elements during acceleration and would be compatible with an average specific gravity of the thoracic contents of about 0.5 since the increase in gradient between the dorsal and ventral recording sites averaged about 0.5 cm. H2O per cm. of vertical distance between the sites per G to which the animal was exposed. Esophageal and pericardial pressures were similar or somewhat less negative than the intrapleural pressure at the same horizontal plane in the thorax. All dogs showed decreases in arterial oxygen saturation during exposures to $6G_{\rm X}$ when breathing air or 99.6% oxygen similar to those previously observed in normal human subjects. Collapse of alveoli and consequent arterial-venous pulmonary shunting of blood appears to be the most likely mechanism for the arterial desaturation observed.

PUBLICATION REVIEW

This technical documentary report is approved.

JOS. M. QUASHNOCK Colonel, USAF, MC

Chief, Biomedical Laboratory

TECHNICS FOR MEASUREMENT OF INTRAPLEUPAL AND PERICARDIAL PRESSURES IN DOGS STUDIED WITHOUT THORACOTOMY AND PETHODS FOR THEIR APPLICATION TO STUDY OF INTRATHOPACIC PRESSURE RELATIONSHIPS DUPING EXPOSURE TO FORWARD ACCELERATION

The project currently being pursued under this contract is an investigation of the mechanisms of the decreases in arterial oxygen saturation and increases in right atrial pressure referenced to mid-chest level, which have been observed in man and in dogs during exposures to forward acceleration in the supine rosition.

In preliminary experiments carried out on four dogs, technics were developed in collaboration with Dr. David E. Donald (D.V.M., Ph.D.) and Dr. A. Clark Molan for positioning without thoracotomy of No. 4F teflon radiopaque catheters so that their tips rested in the pericardial space and in the ventral and dorsal right pleural space at a cross-sectional level approximately at the tricuspid valve. These catheters were fluid-filled and connected to Statham P23D strain gauges. Their length is 40 cm., internal diameter 0.7 mm. and external diameter 1.3 mm. The technic of introduction was designed so that no air was admitted at the puncture sites.

The equipment utilized for recording intrapleural pressures using this percutaneous technic is illustrated in Figure 1.
The arrangement for the percutaneous nuncture through the appropri-

ete intercostal space is illustrated in the middle panel. The P23D Statham strain gauge teflon catheter system is filled with Pinger's solution containing 25 mg. of Webarin per liter. The birdseve catheter tin shown in the top panel is withdrawn inside of the tip of the No. 16 page puncture needlo. The catheter passes through an air-tight rubber masket assembly attached to the hub of

this needle by

ASSEMBLY FOR CATHETER INSERTION
FOR RECORDING OF INTRAPLEURAL PRESSURES
Close-Up of Needle and Catheter Tip

)

Assembly During Puncture

المناخ وسد

Assembly During Pressure Recording

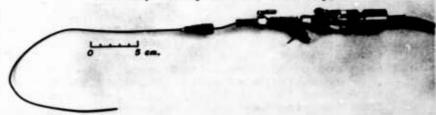


Figure 1. Assembly for percutaneous insertion of a No. 4F catheter for recording of intrapleural pressure. See text for description of technic.

The teflon tubing, from which these catheters were fabricated, was purchased from the ".f. Catheter Company, Glens Falls, New York.

threaded male luer lok hypodermic fitting. The tightness of fit of the rubber gasket surrounding the catheter shaft can be varied by tightening or loosening the internally threaded collar fitting (packing nut) shown in the bottom two panels. After the catheter has been threaded through this assembly which has been previously filled with Ringer's solution the packing nut is tightened with finger pressure so that the tightness of fit to the catheter shaft is such that the catheter can, with reasonable care, just be forced into or withdrawn from the needle without buckling of its shaft.

Dogs under morphine (7.5 mg./kg., subcutaneously) supplemented with sodium pentobarbital anesthesia were used. The hair was clipped from the thorax, the ventral portions of the neck and inguinal regions. A flexible cuffed polyvinyl endotracheal tube was inserted and the animal positioned in the left decubitus position.

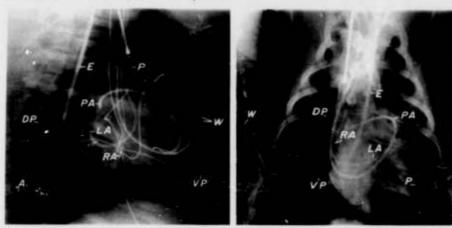


Figure 2. Lateral (left panel) and anteroposterior (right panel) roentgenograms of the thorax of a dog showing positions of catheter tips for recording pressures from multiple sites in the thorax. Catheters were introduced and positioned without thoracotomy using fluoroscopic and pressure monitoring. (A) Tip of catheter in thoracic aorta introduced percutaneously via right femoral artery, (DP) Tip of catheter in potential intrapleural space in right paravertebral gutter, (VP) Tip of catheter in potential right retrosternal intrapleural space, (E) Esophageal catheter, (PA and PA) Tips of catheters introduced percutaneously via left external jugular vein and positioned with tips in the pulmonary artery and right atrium respectively, (IA) Tip of catheter introduced into left atrium via transseptal puncture from the right external jugular vein, (P) Catheter in pericardial sac introduced intramediastinally from a percutaneous puncture cephalad to the suprasternal notch, (W) Wires inserted through lateral cortex of ribs bilaterally for fixation of thistle tube system used for determination of zero pressure reference levels before and during centrifuge rotation.

The needle connected to the ventral pleural catheter assembly was inserted via a 2 mm. stab wound through the skin in the 4th interspace in approximately the mid-axillary line with its tip directed ventrally along the interspace at an angle of about 30 to 45 degrees with the skin surface. The needle was advanced slowly through the intercostal musculature with continuous pressure monitoring until a characteristic sensation transmitted through the needle shaft was perceived when the needle tip perforated the delicate but rather tough parietal pleura. This was followed by the attainment of a negative pressure varying with respiration in a characteristic manner. The needle was then held in this position and the catheter advanced through it under fluoroscopic control. The catheter can be manipulated quite easily in the potential pleural space. Its tip was positioned between the sternum and the ventral surface of the heart (Figure 2)

The dorsal pleural catheter was introduced in a closely similar fashion with the puncture needle directed dorsally along the 5th intercostal space. Its tip was positioned in the right paravertebral gutter dorsal to the heart (Figure 2). After each pleural catheter tip was positioned satisfactorily, a suture was inserted percutaneously so as to pass under the shaft of the puncture needle. This suture was tightened around the needle shaft using a single throw knot and the needle carefully withdrawn over the catheter shaft so as to not disturb the position of the catheter tip. The suture was then snugged tightly around the catheter shaft to prevent any possibility of pneumothorax.

The assembly used for percutaneous introduction of the pericardial catheter is illustrated in Figure 3. The 13 gauge

olive-tipped guide needle used for attaining the position for the pericardial puncture is 26 cm. in length and is equipped with a luer lok gasket packing nut assembly closely similar to that described for the pleural puncture needle. The No. 4F. radiopaque teflon .catheter is introduced into the guide needle through this gasket assembly. This catheter contains a No. 22T gauge-hypodermic tubing stylet. (length: 52 cm.) introduced via a similar air-tight packing nut assembly and connected to a P23D strain gauge

ASSEMBLY FOR CATHETER INSERTION FOR RECORDING OF PERICARDIAL PRESSURES Close-Up Gatheter and Needle Tip for Puncture

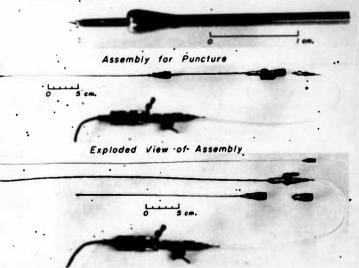


Figure 3. Assembly for percutaneous insertion of a No. 4F catheter for recording of pericardial pressure. See text for description of technic.

manometer via a 30 cm. length of nylon tubing (internal diameter: 1.5 mm., external diameter: 2.1 mm.). This length of
flexible nylon tubing is required to provide adequate mobility
of the puncture assembly. The entire assembly is fluid filled
and the entrapment of minute air bubbles avoided so that an adequate dynamic response for monitoring pressures transmitted through
the stylet assembly is meintained. An "exploded" view of the
complete assembly is shown in the bottom panel of Figure 3.

The tip of the hypodermic stylet used for puncture of the pericardium is carefully positioned so that it protrudes just beyond the birdseye tip of the teflon catheter, as shown in the top panel of Figure 3. The tip of this stylet-catheter assembly is then withdrawn so that it is inside the olive tip of the guide needle.

I dog is positioned in the supine position with his neck extended. A 3 mm. diameter stab wound is made in the skin and underlying fascia just ventral to the trachea and cephalad to the suprasternal notch. The guide needle containing the stylet-catheter assembly is introduced through this stab wound and carefully advanced under fluoroscopic control so that it passes just ventral to the trachea in the mid-line to a position just above the base of the heart. A heavy suture inserted percuteneously and passed under the shaft of the guide needle is snugged down with a single throw-knot to lessen the possibility of passage of air along the needle shaft into the mediastinum. The animal is rotated into the left decubitus position while mainteining the position of the guide needle as nearly unchanged as possible. The guide needle is then advanced under fluoroscopic control until it is seen to indent the cardiac shadow just enterior to the origin of the cephalic great vessels (Figure 2, left panel). The guide needle is then maintained in this position and the styletcatheter assembly advanced through it under fluoroscopic control until the sensation of the needle tip perforating the delicate but tough. pericardium is perceived. The catheter is then advanced over the stylet to the desired position in the pericardial space (Figure 2). The hypodermic stylet is withdrawn until its tip is just beyond the stopcock at the external end of the catheter. This stopcock is then closed, the stylet and packing nut assembly removed and the nylon adapter tubing and strain gauge connected directly to the catheter with care to avoid entrapment of air bubbles at the connection. The stopcock is then opened thus completing the procedure.

Using technics previously developed in the laboratory, catheters which were introduced by percutaneous puncture were positioned with their tips in the pulmonary artery, right atrium, thoracic aorta, iliac artery and left atrium. (WADD mechnical Report 60-634, January, 1961; Circulation Research 19:1196, November, 1961; Proceedings of the Staff Meetings of the Mayo Clinic, 33:536, October, 1958.) Catheters were also positioned with their tips in the esophagus at the level of the tricuspid, and in some animals in the rectum. Pressures transmitted via these catheters and in the endotracheal tube (respiratory airway pressure) were recorded continuously and simultaneously using Statham strain

rauges. The r.p.m. of the centrifuge, the angle of the cockpit from the vertical, and the acceleration at heart level were also recorded.

Thistle tubes for pressure zero reference level corrections during exposure to acceleration were wired to the 4th ribs bilaterally. A-P and lateral roentgenograms of the thorax were taken with a radiopaque grid of known dimensions included in the picture for correction of distortion in subsequent measurements of the vertical distances separating the tips of the pleural pericardial and esophageal catheters and localization of the intravascular recording sites (Figure 4).



Figure 4. Tateral (left panel) and antero-posterior (right panel) roentgenograms of the thorax of a dog showing positions of catheter tips at multiple . thoracic sites following a series of exposures to forward acceleration on a centrifuge. The class thistle tubes (T) fixed by wires to ribs bilaterally and used for zero pressure reference level determinations are also shown. The lead strips (0) which were 'astened to the thistle tubes (left panel) mark the level of the hypothetical coronal plane passing through the mid-anterior posterior level of the thorax. This plane was used as the zero pressure reference level for all intravascular pressure measurements. (A) Tip of catheter in thoracic aorta, (DP) Tip of catheter in potential intrapleural space in right paravertebral gutter, (VP) Tip of catheter in potential right retrosternal pleural space, (E) Esophageal catheter, (PA and RA) Tips of catheters in the pulmonary artery and right atrium respectively, (LA) Tip of catheter in left atrium. The 2 cm. marks indicate the dimensions of reference grids used to correct for and verify the accuracy of the corrections for distortion of the measurements made from these roentgenograms and used to correct the intrapleural and esophageal pressure measurements to pressures at the respective catheter tips.

The animals were fixed in the supine position on a specially designed padded couch which could be tilted around a transverse axis passing approximately through the tricuspid valve. The animal and couch were then transferred to the centrifuge cockpit and the various catheters connected to their respective strain gauge manometers which were positioned as closely as possible to the axis of rotation of the couch. These strain gauges and the thistle tubes were connected to a pressurized wash bottle system filled with heparinized Ringer's solution. This system was used for calibration of the sensitivities of the manometers and establishing the zero reference level of the gauges at mid-A-P chest level.

Correction for the zero shift of the manometers and of the animal during exposures to acceleration was accomplished using the thistle tube system as described in WADD Technical Report 60-634.

The changes in oxygen saturation of systemic arterial blood were recorded continuously by a cuvette oximeter simultaneously with the pressure recordings. Blood was withdrawn through the cuvette by a mechanical constant rate withdrawal syringe via a nylon catheter positioned with its tip in the iliac artery. Sampling was continued for approximately 3 minutes extending over a period including a 15 to 30 second control interval just prior to the exposure, a 60 second exposure to plateau levels of 2, 4, and 66 and continuing for approximately 2 minutes of the recovery period immediately following the exposure.

Simultaneous and continuous recordings were made of the variables mentioned above during 60 second exposures to plateau levels of 2, 4, and 63 with the animal in the horizontal position and breathing air. The zero reference level of each manometer system was recorded after each exposure and a calibration of the sensitivities of all the systems carried out before and after each sequence of 3 exposures. The manometers were opened to the thistle tube system, the menisci in the thistle tubes set to mid-chest level and the zero reference base line of each system recorded continuously along with r.p.m. and cockpit angle during three periods of rotation of the animal and associated transducer assemblies at 2, 4, and 6G.

The physiologic recordings at 6G with the animal in the horizontal position were repeated and then the couch was tilted so that the dog was in the 15° head-up position. The same sequence of exposures, calibrations, and base line recordings were done in this position as carried out previously in the horizontal position. The couch was then tilted so that the dog was in the 15° head-down position and this sequence again repeated. The animal was returned to the horizontal position and a repeat 60 second exposure to 6G carried out.

The pulmonary artery catheter was then attached to a second cuvette oximeter which was in turn connected to the second syringe of the constant-rate withdrawal assembly. The animal was then exposed to 6G for 3 minutes and following a several minute period of recovery the respiratory gas mixture was changed from room air to 99.6% oxygen at ambient pressure. Sixty second exposures to 6G and 4G were carried out followed by a 3 minute exposure to 6G, interspersed with zero reference checks and manometer calibrations as for previous exposures. The absolute accuracy of the cuvette oximeters was checked for each animal by menometric analysis of blood samples withdrawn through both cuvettes while records of oxygen saturation were being made.

Using care not to change the position of the catheters, the animal and couch were removed from the centrifuge cockpit and A-P and lateral roentgenogrems taken of the thorax to verify the position of the various catheter tips (Figure 4).

The animal was then partially exampuinated, his trachea exposed and a lethal dose of sodium pentobarbital given intravenously simultaneously with injection of 50 ml. of formalin into the oral airway with the animal in the 15° head-up position. The trachea was then clamped and the thorax opened carefully to determine the position of the various catheter tips and the gross appearance of the intrathoracic structures. The lungs were removed with care not to allow their collapse and after gross inspection immersed in formalin for subsequent histologic examination.

Experiments on eight dogs have been carried out according to this protocol. In the first four experiments, in addition to the simultaneous recordings of all variables on a two (slow and fast) camera photokymographic assembly, parallel recordings of part of the variables were made on a 7 channel magnetic tape assembly for subsequent electronic data processing. Due to the limitation to 7 channels for magnetic tape recording, it was necessary to repeat the sequence of exposures in each body position in order to obtain magnetic tape records of the variables considered to be of greatest interest.

The last four experiments were carried out without magnetic tape recording and without introduction of the pericardial catheter. The objective of these experiments was to obtain data with as simple and hence as reliable pressure recording assembly as possible and to minimize the chance of inadvertent production of some degree of pneumothorax during the introduction of the pericardial catheter by the suprasternal intramediastinal route used.

Concomitant with these studies of the effects of various levels of acceleration produced on the centrifuge, studies have been carried out at 16 in collaboration with Drs. A. C. Edmundowicz and D. E. Donald. Data obtained showing the relationships of intrapleural pressures at multiple thoracic sites to pericardial, esophageal, and atrial pressures in dogs when under the influence of the normal 16 gravitational field of the earth only are shown in Figures 5, 6 and 7.

The mid-thoracic coronal plane, which is the horizontal plane midway between the ventral and dorsal surfaces of the thorax, was taken as the zero reference level for intravascular pressures. Intrapleural, pericardial and esophageal pressures were expressed as pressures at the catheter tip.

Figure 5 shows representative recordings of the various pressures before and after injections of various volumes of Ringer's solution and air into the pleural space. A general similarity in contour and level of the various intrapleural pressures is evident.

EFFECT OF INTRAPLEURAL INJECTIONS ON INTRAPLEURAL AND RELATED PRESSURES IN SUPINE POSITION (Dog Under Morphine Pentobarbital Anesthesia)

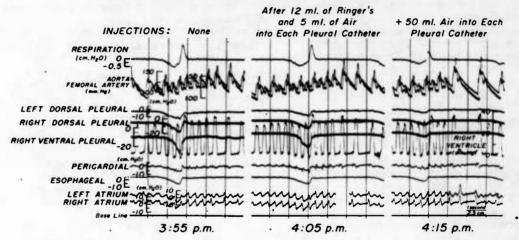


Figure 5. Simultaneous recordings of pressures from multiple sites for study of the interrelationships of intravascular, pleural and other intrathoracic pressures. Note the general similarity in dorsal pleural, esophageal and pericardial pressures which are less negative than the ventral pleural pressure and that injections totaling 17 ml. in volume at each of the three pleural recording sites caused relatively small changes in these pressures.

However, there are systematic differences related to the vertical position of the recording catheter tip in the thorax as shown in Figure 6 which shows 22 observations of intrapleural pressure in 9 mongrel dogs studied in the supine position at 1G. The end-expiratory pleural pressure at the catheter tip is plotted on the abscissa and vertical distance in relation to the mid-coronal plane on the ordinate.

The mean end-expiratory right ventral pleural pressure in these 9 dogs was minus 6.6 cm. of water while the mean end-expiratory

right dorsal pleural pressure recorded simultaneously was minus 0.6 cm. of water. The mean vertical distance between the catheter tips was 11 cm. Thus the mean pleural pressure gradient was approximately 0.5 cm. of water per cm. of vertical distance separating the recording sites.

This evident dependence of pleural pressure on the vertical position of the measurement site suggests that pericardial and esophageal pressures should also be specified in relation to their vertical position in the thorax. Data in this regard are illustrated in Figure 7 in which the simultaneous endexpiratory esophageal and pericardial pressures relative to pleural pressures and right and left atrial pressures in regard to their vertical positions in the thorax of 5 dogs are shown.

VARIATION OF INTRAPLEURAL PRESSURE
WITH VERTICAL POSITION IN THORAX OF 9 DOGS

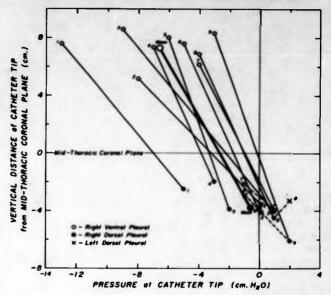


Figure 6. Variation of intrapleural pressure with vertical position of the tip of the recording catheter in the thorax of 9 dogs while in the supine position. Note that the ventral pleural pressure is uniformly more negative than the pressure recorded from more dorsal (dependent) sites in the thorax. The heavy line connects the average values for all dogs. The numerals designate values from individual animals.

Esophageal and pericardial pressures tend to be somewhat higher than the pleural pressures at similar vertical heights in the thorax, though not uniformly so. These data support the suggestion that esophageal and pericardial pressures should also be specified in relation to their vertical position in the thorax.

The belief that the intrapleural pressure gradient is related to the weight of the lungs and other intrathoracic contents is confirmed by the effect of changes in weight on these differences. Data obtained in a dog during the increase in weight produced by an exposure to forward acceleration of 6G are shown in Figure 8. Time, in seconds, is on the abscissa. The bottom stippled area delineates the 6O second exposure to 6G during which time the weight of all bodily structures was increased 6 times.

The top stippled area delineates the pressure difference in cm. of H2O between the ventral and dorsel end-expiratory pleural

pressures. At 1G the ventral pleural pressure was minus 9, the dorsal and the esophageal minus 2 cm. of H₂O.

At 6G the dorsal and esophazeal pressures increased to plus 15 cm. of HoO while the ventral became more negative, minus 20 cm. Ho0: this amounts to a six fold increase in the pressure difference as would be expected if this difference were related to the weight of the intrathoracic structures. The gradient of about 0.5 cm. per cm. of vertical distance per G of acceleration re-

RELATION OF ATRIAL, INTRAPLEURAL AND RELATED PRESSURES TO THE VERTICAL DISTANCE OF THE RECORDING SITE FROM THE MID-THORACIC CORONAL PLANE (Volues for 5 Dogs in Supine Position)

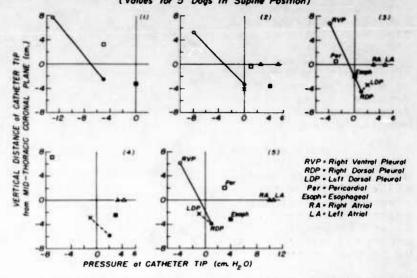


Figure 7. Relation of atrial, intrapleural and related pressures to the vertical distance of the recording site from the mid-thoracic coronal plane. See text for discussion.

mained essentially constant (middle line). It is concluded that, in an animal exposed to the normal 1G gravitation force of the earth, that there are systematic differences in pressure, between the superior and dependent surfaces of the lung, amounting to about 0.5 cm. H₂0 pressure per cm. of vertical distance separating these surfaces.

The magnitude of these end-expiratory pressure differences appears to be directly related to the weight of the intervening lung, its centained blood, and other intrathoracic structures.

The analysis of the data from the series of centrifuge experiments on eight dogs is a formidable task and is, at present, only partially completed. Both manual and electronic data processing methods are being used. Development of the electronic technics and digital computer program is still incomplete so that no firm data from this source are available at the present time.

Circulatory pressures; mean right and left atrial pressures in reference to the mid-dorsal-ventral coronal plane of the thorax were increased to a similar degree during exposures to forward acceleration in the horizontal position and in direct

VARIATIONS IN INTRAPLEURAL PRESSURES WITH CHANGES IN WEIGHT PRODUCED BY FORWARD ACCELERATION

(Dog in Supine Position, Morphine Pentoborbital Anesthesia)

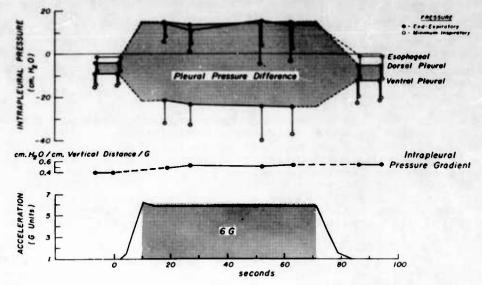


Figure 8. Variations in intrapleural pressures with changes in weight produced by forward acceleration. See text for discussion.

proportion to level of acceleration. The increases in mean pressures amounted to approximately 1 to 2 cm. of H₂O per G increase in acceleration. These changes were somewhat greater in the 15° head-down position and abolished or reversed when in the 15° head-up position. The changes in mean pulmonary artery pressure in reference to mid-chest level were similar. Effects on aortic pressure were more variable, but no remarkable systematic changes were observed.

Intrapleural pressures were referenced to the position of the catheter tip in the thorax determined by A-P and lateral roentgenograms taken prior to and after the series of centrifuge exposures. The degree of negativity of intrapleural pressure in the ventral thorax was uniformly increased during exposure to forward acceleration while intrapleural pressure in the dorsal thorax became positive. As discussed above, these changes are believed to result from the increase in weight of the lungs and other intrathoracic elements during acceleration. The magnitude of the changes would be compatible with an average specific gravity of the thoracic contents of about 0.5 since the increase in pressure gradient between the dorsal and ventral recording sites averaged about 0.5 cm. of H₂O per cm. of vertical distance between the sites per G of acceleration to which the animal was

exposed. The degree of increase in negativity of ventral pleural pressures during exposures was somewhat greater in the 150 headup and less in the 150 head-down than in the horizontal position. The typical changes in intrapleural and esophageal pressures during an exposure to a forward acceleration of 60 in the horizontal position are illustrated in Figure 8.

Esophageal and pericardial pressures were similar or somewhat less negative than the intrapleural pressure at the same horizontal plane in the thorax. The changes in these pressures during acceleration appear to be closely similar to the changes in intrapleural pressure in the same horizontal plane (Figure 8).

All of these dogs showed decreases in arterial oxygen saturation during exposure to forward acceleration of 60 when breathing air or 99.6% oxygen similar to those previously observed in normal human subjects. The severity of the decreases increased with the level of acceleration and although reduced were not prevented by breathing 99.6% oxygen. The decreases in arterial oxygen saturation were of approximately similar magnitude in the horizontal, 150 head-up and 150 head-down positions although in some animals these changes were somewhat greater in the head-down position.

At the termination of the series of centrifuge exposures in two animals 100 ml. of air was injected into the right pleural space via the ventral pleural catheter and a 1 minute exposure to 6G repeated. The presence of this degree of pneumothorax did not apparently alter the effects of an exposure to 6G on the variables under study.

Gross and microscopic examination of the lungs of the animals studied on the centrifuge showed collapse of the alveoli in dependent portions of the lobes and gross overdistention of the alveoli (so as to be visible to the naked eye) in the superior margins of the lungs.

Collapse of alveoli and consequent arterial-venous pulmonary shunting of blood appears to be the most likely mechanism for the arterial desaturation observed.

High negative pressures produced in extra-alveolar portions of the superior portions of the thorax are believed responsible for the emphysematous changes in the superior margins of the lungs and also to be the cause of the acutely incapacitating mediastinal emphysema which developed in a healthy subject during an exposure to 5.5G.

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